Amendments to the Specification:

Please replace the paragraph beginning at page 25, line 24 (paragraph 105 in the published version of the US application), with the following rewritten paragraph:

-- A determination of cell viability and growth incorporated into a matrix material primarily composed of isolated basement membrane is included in one embodiment of the invention. Experimentally, it was previously shown that a matrix material primarily composed of fibrin successfully entraps cells and that these cells were also able to release viral proteins. An experimental study was conducted to determine if the matrix Matrigel™ was able to behave ~~behavior~~ in a similar fashion as fibrin. Matrigel™, as previously discussed, is an isolated basement membrane obtained from ~~for~~ cells cultured *in vitro*, which has been used in a wide variety of *in vivo* and *in vitro* studies of cell attachment, cell growth and angiogenesis. Like fibrin, Matrigel™ is a naturally occurring matrix derived from basement membrane, that has a wide variety of binding sites for cells and factors (including growth factors and cytokines). Matrigel™ has been used extensively as a cell matrix/depot in a wide variety of *in vitro* and *in vivo* studies particularly in the area of tumor cell biology and angiogenesis. Matrigel™ is a liquid at 4°C but becomes a solid biological matrix when warmed to 37°C. This ability to convert Matrigel™ from a liquid to a solid by simply raising the temperature, allows for a wide variety of strategies for entrapping genetically engineered cells, factors proteins and genes. Matrigel™ and other isolated basement membrane materials possess the characteristics to serve as a tissue interactive biological matrix for the ATS. DF-1 cells were utilized to determine the virus and VEGF expression in the DF-1 cell lines. Briefly, Matrigel™ and DF-1 cells (e. g. DF-1, GFP:DF-1, VEGF:DF-1) were mixed together at a ratio of 3:2 and 50 µl of Matrigel™ cell suspension was pipetted into 6-well tissue culture plate. Wells were supplemented with culture media. An aliquot was taken out of each well daily for a total of 1 week and replaced with fresh DF-1 culture media. Aliquots were stored at –70°C till assayed by ELISA for p27 and VEGF expression as described earlier. --

Please replace the paragraph beginning at page 59, line 4 (paragraph 167 in the published version of the US application), with the following rewritten paragraph:

-- To evaluate sensor function in our control mice and Matrigel™ treated mice, sensor function in the mouse was analyzed for between 1 HPI and 6 DPI. In these studies, glucose blood levels parallel sensor function at both 1 and 5 hours post sensor implantation. Analysis of blood glucose and sensor function at 1-day post sensor implantation clearly indicated that although the blood glucose levels were elevated by i. p. injection of the glucose, that there was little or no response from the implanted sensors in the control mouse. Analysis of the blood glucose and sensor function at 2, 3, and 6 days post sensor implantation consistently demonstrated that although blood glucose levels were elevated by i. p. injection of glucose, there was no sensor function in the control mouse as shown in Figure 38. However, glucose sensor functionality in Matrigel™ glucose sensor treated mouse paralleled blood glucose levels. This behavior was consistent for time-points 1 HPI, 5 HPI, 1 DPI, 2 DPI ~~2DPI~~ and 3 DPI. Sensor sensitivity was in the range of under 10 to about 13 nA/mM ~~close to zero~~ for Matrigel™ /sensor treated mouse at 1 DPI, 2 DPI and 3 DPI, as shown in Figure 38. Thus, an ATS, such as Matrigel™, around a glucose sensor increases the lifetime of an implantable glucose sensor. –

Please replace the paragraph beginning at page 59, line 29 (paragraph 169 in the published version of the US application), with the following rewritten paragraph:

-- Evaluation of sensor function in the mouse model was accomplished by analyzing sensor function in control mice and in the dexamethasone treated mice for 1 HPI and up to 8 DPI. In these studies, glucose blood levels parallel sensor function was monitored at both 1 and 5 hours post sensor implantation, as shown in Figure 39 (4 hr and 8 hr) ~~(1hr and 5hr)~~, for both control mice, having no dexamethasone treatment, and dexamethasone treated mice. Analysis of the blood glucose and sensor function at

1day post sensor implantation in the control mouse clearly indicated that although the blood glucose levels were elevated by i. p. injection of the glucose, that there was little or no response from the implanted sensors in the control mice as shown in Figure 39 ~~38~~ (1d). However, analysis of the dexamethasone mouse showed glucose levels paralleled sensor function. Analysis of the blood glucose and sensor function at 3, and 8 days post sensor implantation in the control mouse consistently demonstrated that although blood glucose levels were elevated by i. p. injection of glucose, there was no sensor function as shown in Figure 39 ~~38~~ (3d and 8d). This rapid loss of sensor sensitivity and sensor function within the first day post implantation was seen in virtually all sensors tested in the control mouse model. Furthermore, the general pattern of loss of sensitivity and sensor function seen in our control mice was similar to that seen in both other animal models and man. On the other hand, glucose sensors implanted into mice treated with dexamethasone i. p. showed blood glucose levels paralleling sensor functionality. Thus, by controlling inflammation around an implanted glucose sensor it is possible to extend the lifetime and functionality of a glucose sensor. This experimentally demonstrates that systemic administration of dexamethasone dramatically enhance the function of a glucose sensor when implanted in the skin of mice. --